THE PLANT DISEASE REPORTER

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PLANT DISEASE EPIDEMICS
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IDENTIFICATION SECTION

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PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION

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RECENT DISCOVERY OF HOJA BLANCA, A NEW RICE DISEASE IN FLORIDA, AND VARIETAL RESISTANCE TESTS IN CUBA AND VENEZUELA

John G. Atkins and C. Roy Adair[1]

Summary

Hoja blanca, a potentially destructive new rice disease, was found near Belle Glade, Florida, in September 1957. Previously the disease had been observed and studied in Cuba and Venezuela where it has caused serious losses. The symptoms are similar to the symptoms described for the stripe disease in Japan. It is thought that the causal agent is an insect-transmitted virus. During 1957 a large number of varieties and selections were grown in Cuba and Venezuela. A large number of varieties of the japonica or short-grain type were resistant. All of the United States long-grain varieties and the commonly grown short-grain varieties were susceptible. However, several minor United States short- and medium-grain varieties and selection such as Colusa, Asahi, Lacrosse, and Missouri R-500 were resistant, and Arkrose was moderately resistant.

Hoja blanca, a potentially serious new disease of rice, was recently found for the first time in the United States near Belle Glade, Florida. Symptoms suggestive of hoja blanca were first observed in late August 1957, and definitely identified on September 12. A USDA News Release announcing the discovery of the disease in Florida was issued on September 18. Previously the disease was known from Cuba, Venezuela, Panama, and Costa Rica.

The name of the disease, "hoja blanca", is a Spanish name meaning "white leaf". The disease has been present in Cuba since at least 1954, according to available information. By 1956 it was widespread there and caused severe yield losses of the commonly grown United States varieties, such as Bluebonnet 50 and Century Patna 231, that are used almost exclusively for rice production in Cuba. Hoja blanca appeared in Venezuela in 1956, and caused serious losses in certain rice areas, particularly in the State of Portuguesa. The disease has spread rather rapidly and is probably present in other South or Central American rice-growing areas. In Cuba and Venezuela the writers have estimated yield reductions of as much as 75 percent in affected fields. According to reliable reports some fields were so severely damaged that they were not harvested.

SYMPTOMS

The first symptoms are one or more white stripes on the leaf blade, or the entire leaf blade may be white, or the leaves may be mottled in a typical mosaic pattern. Figures 1 and 2 illustrate the leaf symptoms. Diseased plants are reduced in height, and in severely affected fields the panicles of healthy plants are taller than those of diseased plants. The panicles of diseased plants are somewhat reduced in size and often are not fully exserted from the sheath. The lemma and palea show a brownish discoloration and dry out rapidly, and frequently are distorted in shape. The floral parts are often absent, or if present they are sterile. As a result, panicles of diseased plants contain few or no seeds and remain in an upright position. Infected plants are not killed by the disease, and new tillers of a second or ratoon crop often show no symptoms. Both normal and diseased tillers frequently are observed on the same plant.

Several grasses growing in or adjacent to fields of diseased rice often show symptoms similar to those of hoja blanca on rice. Echinochloa colonum and Panicum fasciculatum have shown such symptoms in Florida, as well as in the other countries. Malaguti (3) and Malaguti, Diaz and Angeles (4) in Venezuela, and Mukoo and Iida (5) in Cuba have reported grasses with similar symptoms.

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Table 1. Tabulation of resistant entries in Hoja Blanca Nursery No. 1 by country of origin, grown in Cuba and Venezuela, 1957.

Country of origin	Number entries tested	Number classed as: Resistant	Moderately resistant
United States	831	48	29
China	528	135	77
Japan	290	182	45
Korea	105	50	32
Taiwan	49	45	1
Burma	5	0	0
India	29	2	2
Indonesia	10	0	0
Philippines	5	0	0
Thailand	4	1	0
Argentina	11	6	0
Brazil	12	3	0
Br. Guiana	3	0	0
Chile	2	2	0
Peru	16	1	2
Venezuela	16	2	0
Costa Rica	4	2	2
El Salvador	15	2	2
Haiti	15	1	0
Jamaica	2	1	1
Austria	2	0	1
France	11	6	1
Greece	2	0	1
Italy	31	15	4
Portugal	17	13	1
Spain	10	9	0
Yugoslavia	3	1	0
Iran	7	1	0
Turkey	26	7	6
Africa, except Egypt	15	0	0
Egypt	6	3	3
Miscellaneous	8	2	0
"Check varieties"	110	0	0
Totals	**2200**	540	210

Table 2. Hoja blanca readings for certain resistant varieties and selections along with the reaction of leading United States rice varieties in Cuba and Venezuela, 1957.

Variety or selection	C. I. number	Hoja Blanca Ratings[a]				Probable reaction
		Cuba		Venezuela		
		May	July	May	August	
Fortuna	1344	3	8	7	9	S
Rexoro	1779	2	7	8	8	S
Zenith	7787	3	8	5	8	S
Magnolia	8318	2	7	8	8	S
Texas Patna	8321	2	7	6	8	S
Bluebonnet 50	8990	2	7	7	9	S
Century Patna 231	8993	3	5	7	8	S
Nato	8998	3	6	6	9	S
Toro	9013	3	8	6	9	S
Colusa	1600	0	0	0	--	R
Arkrose	8310	2	1	3	tr	MR
Asahi	8312	tr	0	0	0	R
Lacrosse	8985	0	0	1	0	R
Lacrosse x Magnolia	9001	2	0	0	0	R
Missouri R-500	9155	1	0	1	0	R
Bruin Sel. x Zenith	9209	0	0	tr	0	R
Hgb. Mix. 11-49-19-5	9368	0	0	0	0	R
B53-2604, B438A Rogue	9369	0	0	0	0	R

[a]Visual rating scale: 0-9 -- 0 = No disease. 9 = Severe disease.

The writers have noted that the symptoms of hoja blanca and those described for the disease known as the stripe disease (Oryza Virus 2) in Japan are similar. Mukoo and Iida (5) as well as Malaguti and co-workers (3, 4) also have pointed out this similarity. The illustration of the stripe disease given by Kawada et al. (2) indicates further that at least certain symptoms of the two diseases are similar or identical. Because of the nature of the symptoms of hoja blanca and their similarity to those of the stripe disease, it has been assumed that, like stripe disease, hoja blanca also is caused by a virus that probably is transmitted by insects. In Venezuela, Malaguti, Diaz and Angeles (4) have reported transmission of hoja blanca by leafhoppers (at present not otherwise identified).

VARIETAL REACTION

On the basis of surveys made in Cuba and Venezuela in late 1956 and early 1957 (1), it was concluded that hoja blanca was a serious rice disease in those countries and that it constituted a threat to the rice industry of the United States. In 1957 the reaction of a large number of United States rice varieties, selections, and introductions from the United States Department of Agriculture world rice collection was determined in tests conducted in Cuba and Venezuela[2]. Hoja blanca Nursery No. 1, consisting of 2200 entries, was sown on two dates near Acariqua, Venezuela. Ratings were made on the two plantings approximately 100 and 95 days, respectively, after seeding. The nursery of 2200 entries and an additional nursery of 1725 entries were grown in a test area near Jobabo, Cuba, where observations were made at approximately 40 and 95 days after seeding. Certain United States varieties were included every 20 rows as "check" varieties throughout the nurseries. Rice selections and varieties included in these tests consisted of diverse types from several countries of the world. Natural infection was

[2]The writers acknowledge the assistance and cooperation of Mr. William C. Davis, Vice President, Arrozal Bartes, S. A., Jobabo, Cuba; and Mr. Rufus K. Walker, Research Director, Narfarm, Valencia, Venezuela (Superintendent, Rice Experiment Station, Crowley, Louisiana, on leave of absence).

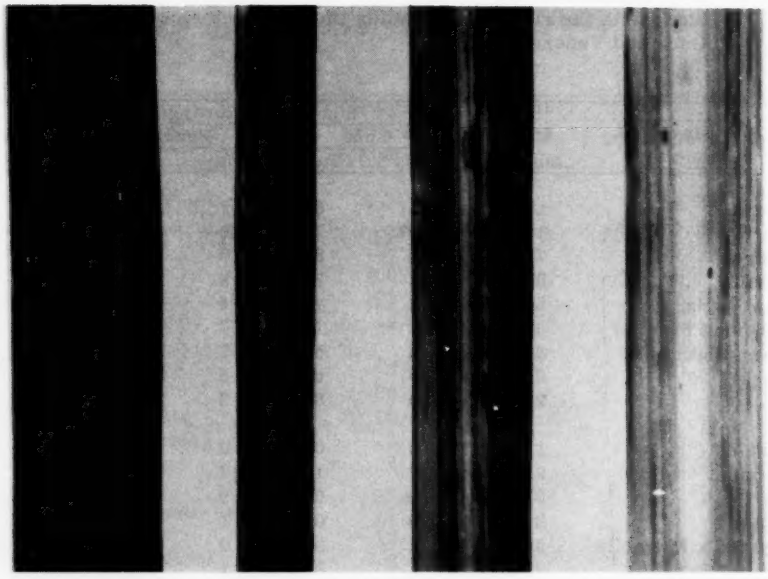

FIGURE 1. Leaf symptoms of hoja blanca. Normal leaf at left.

FIGURE 2. Typical appearance of hoja blanca in the field.

(NOTE: The dark leaf spots, caused by Helminthosporium oryzae, in the figures represent a separate disease.)

Photographs by Henry M. Spelman, III, Everglades Experiment Station, Belle Glade, Florida.

heavy on susceptible entries at both locations. A scale of 0 to 9 was used for rating severity.

A fairly large number of the entries were rated as resistant to hoja blanca, since they showed few or no foliar symptoms and growth and grain development were normal. A large number of the resistant entries listed in Table 1 came from Japan, China, Taiwan, or Korea. Most of the resistant entries are japonica types, which may be characterized as short-grain types with rough, fairly narrow, dark green leaves. Varieties from other countries were of this type, which indicates Japanese origin. Probably, if the origin of all the resistant entries included in the tabulation were known, the total number of resistant varieties listed from Japan and, perhaps, China would be larger, or else there would be many duplications. Included among the resistant United States rices in the tabulation are several that derived their resistance from foreign varieties. Many of the entries listed as resistant are undesirable types because of agronomic or grain characteristics or of susceptibility to blast (Piricularia oryzae).

A few of the United States short- and medium-grain varieties of minor importance, and several selections, were found to be resistant. However, all of the more widely grown United States rice varieties were susceptible. The one possible exception is Colusa, which was resistant. No long-grain varieties were found to be resistant to hoja blanca. Readings for the most important rice varieties of the southern rice area are given in Table 2. All of these were definitely quite susceptible. Caloro, the leading California variety, also was susceptible. A few types that were consistently resistant in all tests are listed in Table 2, together with their disease ratings for comparison with ratings for the susceptible varieties. Some of the resistant selections are adapted types and, if necessary, they could be grown in the United States.

One of the objectives of the 1957 tests was to screen the United States Department of Agriculture rice collection for sources of hoja blanca resistance. As shown in Table 1, a rather large number of entries which might be used in a breeding program to develop hoja blanca resistant varieties is available. The very few long-grain varieties that were rated as resistant are of undesirable plant types. A check as to the origin or parentage of the resistant United States varieties and selections indicates that japonica or short-grain types are the sources of their resistance. Asahi is an introduced variety from Japan, while Colusa was selected from an introduction from Italy that originally came from China. Possibly the resistance of Lacrosse, Missouri R-500, and certain other selections came from short-grain parents. Selections with Colusa as a backcross parent were generally resistant. A number of resistant selections had Lacrosse as one of the parents. Some of the selections from each of two crosses between Lacrosse and a susceptible parent were resistant like Lacrosse, whereas others were completely susceptible. These results indicate that it should be possible to obtain resistant strains of all grain types.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE.

DIFFERENTIAL RESPONSE OF DWARF BUNT TO SEED AND SOIL SURFACE TREATMENT WITH HEXACHLOROBENZENE[1]

Laurence H. Purdy[2]

Dwarf bunt (Tilletia contraversa Kuehn) in winter wheat occurs sporadically in almost all of the wheat-growing areas of the Pacific Northwest. Its occurrence has assumed a cyclic pattern in which heavy infections occur in alternate years. Since dwarf bunt infests the soil, this cycle has been attributed to cultural routines that determine when the inoculum is at or near the soil surface (4). Accumulated evidence suggests that infection of wheat by the dwarf bunt organism takes place at or very near the soil surface (7). This may explain why dwarf bunt is not controlled by seed treatment, since the fungicide placed on the seed apparently is too far removed from the infection site to give protection.

In the Pacific Northwest seed treatment fails to protect wheat from infection by the dwarf bunt fungus. Because of this fact, and since there has been no other acceptable chemical control measure, the use of resistant varieties has been the sole means of combatting this disease. However, through the development of new races the dwarf bunt organism is capable of attacking varieties possessing all major types of bunt resistance. Thus, other means of control are essential.

Experiments have shown that in-the-furrow subsurface applications of mercury fungicides gave no protection against dwarf bunt, while hexachlorobenzene (HCB) gave partial control under these same conditions (5).

Dwarf bunt control obtained from the application of fungicides to the surface of the soil in Japan (6), Europe (1, 2, 9, 10), and Utah (8) demonstrates the effectiveness of this type of soil treatment. This is a recommended practice in the dwarf-bunt infested areas of Europe (2). In addition to soil treatments, the continued use of adequate seed treatment with appropriate materials is considered indispensable by European workers (2) and its value in Canada was also demonstrated by Fushtey (3).

Because of the good control obtained from soil-surface applications of fungicides in other infested areas, and the need for a more thorough evaluation of seed treatment for dwarf bunt control, experiments were designed to determine the effectiveness of HCB against dwarf bunt as a soil-surface treatment and as a seed treatment in the Pacific Northwest.

MATERIALS AND METHODS

In the soil-surface-treatment experiments the winter wheat variety Orin was sown 1/2 inch below the soil surface in open furrows in soil naturally infested with dwarf bunt. In one-half of the furrows additional inoculum was added by spraying the surface of the soil after sowing with a suspension of dwarf bunt spores. The seed sown in these same furrows was also inoculated with dwarf bunt spores. The remaining naturally infested furrows were sown with untreated bunt-free seed and no additional inoculum was added to the soil. Sanocide (40% HCB) was applied to the surface of the soil at the rate of 10 pounds per acre, on four different dates. The first application was at sowing, the second at emergence, the third 4 weeks after emergence, and the fourth in the early spring. Each treatment was replicated five times.

The effectiveness of HCB as a seed treatment for dwarf bunt control was determined by the application of Sanocide to the seed of Orin wheat at the rate of 2 ounces per bushel, which is twice the rate recommended for the control of soil-borne common bunt. The seed was sown at three depths, 1/2, 1, and 3 inches, in soil that had been sprayed previously with a suspension of dwarf bunt spores. There were five replications of each planting depth.

The percentages of dwarf bunt were based on the total number of heads per row.

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[1] Cooperative investigations of the United States Department of Agriculture, Agricultural Research Service, Crops Research Division, and the Agricultural Experiment Stations of Idaho, Oregon, and Washington.

[2] Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Pullman, Washington. Scientific Paper No. 1658, Washington Agricultural Experiment Stations, Pullman, Washington.

Table 1. Effect of time of application of HCB to the soil surface on the control of dwarf bunt.

Time of application	Percentage of dwarf bunt	
	Inoculated soil	Naturally-infested soil
Check (no treatment)	38.0	8.0
At sowing	5.0	0
At emergence	2.0	0
4 weeks after emergence	0.5	0
Spring	20.0	5.0

Table 2. Effectiveness of HCB seed treatment in controlling dwarf bunt at different sowing depths

Sowing depth (inches)	Percentage of dwarf bunt	
	Untreated seed	HCB treated seed
1/2	41	45
1	24	23
3	5	6

RESULTS

The results of the soil-surface treatments are summarized in Table 1.

The check rows had 38 and 8 percent dwarf bunt, respectively, in inoculated and naturally infested soil. Good control was obtained from the first three applications in both inoculated and naturally infested soil. In the inoculated soil best control was obtained with the applications made 4 weeks after emergence. No control resulted from the spring application (early March) and this failure suggests that infection had apparently taken place before the time of application.

The amount of dwarf bunt that developed in the tests to evaluate HCB seed treatment (Table 2) showed a definite relationship to the depth of planting, as pointed out by Meiners, Kendrick, and Holton (7). The greatest amount occurred in the shallow sowing, and the least in the deep planting. At all sowing depths seed treatment with HCB failed to control dwarf bunt.

These results suggest that soil-surface applications of HCB can be used in localized areas of heavy dwarf bunt infestation to provide adequate protection from infection. The continued use of seed treatment with appropriate chemicals is also a necessary practice to reduce or minimize the spread of individual races of dwarf bunt to uncontaminated areas.

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UNITED STATES DEPARTMENT OF AGRICULTURE, AGRICULTURAL RESEARCH SERVICE, CROPS RESEARCH DIVISION; AND IDAHO, OREGON, AND WASHINGTON AGRICULTURAL EXPERIMENT STATIONS

EFFECTS OF SOME MATERIALS ON STEWART'S BACTERIAL WILT OF SWEET CORN WHEN APPLIED AS SEED TREATMENTS[1]

Lansing E. Williams

Summary

Streptomycin, terramycin, tetracycline, indoleacetic acid, 2,4,5-trichlorophenoxyacetic acid, and sodium borate applied in water solutions or suspensions to Golden Bantam sweet corn seeds reduced the severity of Stewart's bacterial wilt on subsequent seedlings. Generally, the most effective concentrations in reducing wilt produced phytotoxic symptoms on the seedlings.

Stewart's bacterial wilt of sweet corn is a serious disease, particularly if the young susceptible seedlings become affected. Resistant varieties as well as insecticidal sprays to control the insect vector have given a measure of control and have been recommended (4, 6, 8). In addition, foliar applications of antibiotics and surface-active agents have been found to reduce the severity and incidence of wilt (1, 3, 7).

During a study of the effect of foliar applications of antibiotics and surface-active agents on the severity of Stewart's wilt, it was found that seedlings arising from seeds soaked in streptomycin solutions exhibited a degree of resistance to wilt. Subsequently, several different compounds were screened for their effect on Stewart's wilt when applied as seed treatments. A material effective as a seed application would have economic significance due to the low rate of treatment per acre and the protection given during the susceptible early seedling stages when large losses in stands may occur.

MATERIALS AND METHODS

Golden Bantam sweet corn seeds were soaked 20 to 24 hours in water solutions or suspensions of the test compounds, then removed, drained, and planted in soil in wooden flats in the greenhouse at approximately 28° C. Seeds soaked in water served as controls. When the subsequent seedlings were in the two- to three-leaf stage they were inoculated by clipping the plants at the level of the coleoptile and spraying the stubs with a water-suspension of bacterial cells (2). The bacterial cells were removed from 2- to 3-day-old yeast extract-agar slants of Bacterium stewarti E. F. Sm., suspended in distilled water, and the resulting suspension adjusted with an electrophotometer so that light transmission was reduced 15 percent. Approximately 1 week after inoculation and formation of new foliage the severity of symptoms was estimated. Symptoms were rated on a scale of 0 to 5, where 0 represented a healthy and 5 a dead plant. Each treatment was replicated five times in separate flats with a control row included in each individual flat.

RESULTS

Of the materials tested, six gave significant control: sodium borate (borax), indoleacetic acid (IAA), 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), terramycin, streptomycin, and tetracycline[2]. These results were confirmed in two subsequent experiments; the results of the third experiment are presented in Table 1.

All of the effective materials induced phytotoxic symptoms in the host plants, particularly at the higher concentrations. The three antibiotics induced considerable chlorosis of plants as well as stunting; at the higher concentrations a few plants were completely chlorotic with no evidence of chlorophyll formation. In addition to chlorosis and stunting terramycin also induced "leaf-stringing". Sodium borate did not induce a complete chlorosis but instead a chlorotic-streaking of seedling leaves. The phytotoxic effects of 2,4,5-T were primarily

[1] Published with approval of the Director of the Ohio Agricultural Experiment Station as Journal Article No. 80-57.

[2] Streptomycin STS (54% streptomycin base) supplied by Merck and Co., Inc.; soluble terramycin (21% terramycin base) supplied by Chas. Pfizer & Co., Inc.; 2,4,5-T supplied by Monsanto Chem. Corp; and tetracycline HCl supplied by Bristol Laboratories.

Table 1. Severity of bacterial wilt in sweet corn seedlings from seeds soaked in water solutions of indicated chemical compounds.

Material	Concentration ppm	Index of[a] disease severity	Percent of plants diseased
Streptomycin	500	0.16**	17**
	250	0.28**	27**
	125	0.36*	34*
	62	0.45	45
Control	-	0.71	55
Tetracycline	2000	0.00**	00**
	1000	0.10**	19**
	500	0.25**	31**
	100	0.82	57
Control	-	0.91	68
Terramycin	500	0.08**	9**
	250	0.24**	27**
	125	0.68	49
	62	0.67	51
Control	-	0.67	49
IAA	2000	0.02**	8**
	1000	0.18**	14**
	750	0.50*	34*
	500	0.50*	32*
Control	-	0.80	58
2,4,5-T	500	0.08**	9**
	250	0.24**	27**
	125	0.63	49
	62	0.67	51
Control	-	0.67	49
Sodium Borate	2000	0.26**	24**
	1000	0.28**	22**
	750	0.64**	45*
	500	0.98	56
Control	-	1.01	63

** Statistically different from the control at the 0.01 level.
* Statistically different from the control at the 0.05 level.
[a] Derived as the mean of the ratings of plants arising from 5 replications of 20 seeds each.

Table 2. Effect of indicated seed treatments on sweet corn stands.

Material	Concentration ppm	Number[a] of living plants	Condition of plants
Control	--	80	
Streptomycin	500	65**	Approximately 90% size of controls; 5 to 10% exhibiting chlorotic streaks.
	250	69*	Very slight stunting; 1 to 5% exhibiting chlorotic streaks.
	125	73	Very slight stunting.
Tetracycline	1000	22**	Approximately 40% as large as controls.
	500	40**	Approximately 60% as large as controls.
Terramycin	500	3**	Approximately 20% as large as controls.
	250	17**	Approximately 40% as large as controls.
	125	36**	Approximately 75% as large as controls.
IAA	1000	67*	Approximately 75% as large as controls.
	500	75	No apparent phytotoxicity.
2,4,5-T	500	2**	Approximately 10% as large as controls.
	250	12**	Approximately 40% as large as controls.
Sodium borate	1000	65**	No apparent phytotoxicity.
	750	69*	No apparent phytotoxicity.
	500	73	No apparent phytotoxicity.

**Statistically different from the control at the 0.01 level.
*Statistically different from the control at the 0.05 level.
[a]Derived as the mean number per row from 4 rows planted of 100 seeds each.

manifested in a large reduction of seed germination and stunting of seedlings. IAA produced no evident phytotoxic effects in the seedlings, with the possible exception of a slight stunting effect.

To determine the phytotoxic effects of these compounds on plant survival, seeds were treated and planted in the field. Four replications of 100 seeds each were planted in randomized plots. Stands and phytotoxic symptoms were observed and recorded approximately 45 days after planting. These data are presented in Table 2. Due to low incidence, no disease data were taken.

All treatments except the lowest levels of sodium borate and IAA resulted in decreased stands relative to the control. These stand losses due to phytotoxicity may have been greater than indicated if more young plants died from wilt in the control rows than died in the other treatments. The low incidence of wilt in the more mature plants would indicate that few young plants died from this cause.

DISCUSSION

While this work was in progress, Rich reported that 2,4,5-T, streptomycin, terramycin, captan, and Crag 1 were effective against Stewart's wilt when applied as seed treatments (5). In Rich's tests indoleacetic acid at 50 ppm gave no control; in these tests it was found that a concentration of 500 ppm was necessary for a significant reduction in wilt. Salicylic acid, penicillin, and captan were other compounds common to both studies. The first two compounds were inactive in both studies; whereas Rich found slight activity with captan.

As mechanical harvesting and processing of seed corn causes a high percentage of injuries to seed coats, the seed lot used in this type of study would affect the entrance and subsequent action of seed treatment materials and give rise to some differences in results from

test to test. This also may explain why some seedlings from seeds soaked in certain solutions of the antibiotics were entirely chlorotic whereas others from seed soaked in the same solutions were only slightly affected. In general, the more chlorotic the seedling the greater its resistance to wilt. Whether the greater resistance to wilt is due to a higher concentration of test material in the chlorotic host plants or is in some way related to the chlorosis is not known.

Due to the accompanying phytotoxicity at disease-controlling concentrations, the use of these materials in their present form as seed treatment protectives for Stewart's wilt does not appear too promising. But it is significant that materials have been found which will enter seeds and move into or affect subsequent seedlings and protect them against wilt. Perhaps new or similar materials or different formulations of these same materials may be found which will increase disease control or decrease phytotoxicity. Because of the ease of treatment and the low rate of application per acre this type of protection is very desirable.

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RESISTANCE OF SOYBEANS TO THE SOYBEAN CYST NEMATODE AS DETERMINED BY A DOUBLE-ROW METHOD

J. P. Ross and C. A. Brim[1]

The soybean cyst nematode (Heterodera glycines Ichinohe) has been found in North Carolina, Tennessee, Missouri, Arkansas, Kentucky and Mississippi. Since this pathogen threatens soybean production in infested areas, resistance to it would be of great value to growers.

During the spring of 1957 approximately 2800 selections and varieties of soybean (Glycine max) were evaluated for resistance by means of a double-row planting method.

One of the greatest difficulties with field evaluations of resistance is the lack of uniformity in nematode population throughout the planting area. This handicap was overcome by planting two-row plots consisting of a standard susceptible line and an entry being evaluated. The infested field was 120 by 450 feet. Seed of each test entry were planted in 5 feet of row with a 1-foot space between entries. After the entire field had been planted with the test lines in rows 32 inches apart, seed from a glabrous line of soybean[2] previously found to be highly susceptible to the soybean cyst nematode were planted in rows approximately 6 inches to one side of each row of the test lines.

White cyst[3] indices for each entry were recorded 1 month after planting when the white cysts attached to the roots were visible. At least six plants from each entry were dug and the roots washed, and each root system was given a rating on the basis of the number of cysts present. If a test strain contained less than 10 cysts per root system, roots of the adjacent susceptible check plants were similarly examined for cysts. The check plants provided a good indication of the nematode population in the proximity of the test strain. The glabrous character of the susceptible check readily distinguished it from the test strains. If an entry received a lower score than the check, resistance was indicated. Many lines were observed to have low ratings, but when the roots of the adjacent susceptible check plants were examined it was evident that the nematode population in that particular area was very low.

Eight of the entries tested in this manner appeared to have high resistance. These eight entries were evaluated again in the field, by the same method. Results of both evaluations, including the susceptible check ratings, are presented in Table 1. It is apparent from these results that in those cases where the check rating was low, indicating a low nematode population, the rating obtained for the test entry was erroneously low (viz. P.I. 189920 and 189969). However, in the cases where the check rating was high, indicating a high nematode population, the rating given the test entry was substantiated in the second evaluation.

The double-row method used in these evaluations could also be used for evaluating other crops for nematode resistance. The method might also find application in screening plants for resistance to other diseases caused by soil-inhabiting pathogens, such as root rots, wilts and foot rots.

The results indicate that resistance to the soybean cyst nematode is available within our present soybean germ plasm. Ichinohe and Asai[4] report that two resistant soybean varieties, "Daiichi-hienuki" and "Nanguntake-date", show almost no symptoms of the disease and allow fewer larvae to mature than susceptible varieties. These two varieties were included in our evaluations and both favored the development of large numbers of female nematodes. The existence of biological races within Heterodera glycines might explain this discrepancy.

Roots of the resistant lines listed in Table 1 are invaded by second-stage larvae, but the number of larvae reaching maturity was found to be very small, and in most cases larvae do not mature. Further investigations on the host-parasite relationships of the resistant lines are in progress.

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[2] Supplied by E. E. Hartwig, Delta Branch Experiment Station, Stoneville, Mississippi.

[3] "White cyst" as used in this paper is understood to mean the living adult female of the soybean cyst nematode. It does not include the true cyst which is formed from the body of the dead female.

[4] Ichinohe, M. and K. Asai. 1956. Studies on the resistance of soybean plants to the nematode, Heterodera glycines. I. Hokkaido National Agricultural Experiment Station Research Bulletin. Sept. 71: 67-79.

Table 1. White cyst indices for eight entries of soybeans evaluated for resistance to the soybean cyst nematode, Heterodera glycines, in double-row plots.

Entry	Maturity Group	First Evaluation		Second Evaluation	
		Entry	Check	Entry	Check
Ilsoy	III	1,1,1,2,1,1	4,4,4,4	1,1,1,2,2,1	4,4,4,4
Peking	III	1,1,1,1,2,1	4,4,4,4	1,1,1,2,1,1	4,4,4,4
P.I. 189920	III	2,1,1,2,1,1	2,2,2,2	4,4,4,4,4,4	4,4,4,4
P.I. 189969	III	1,1,1,1,2,2	2,2,2,2	4,4,4,4,4,4	4,4,4,4
P.I. 79693	III	3,2,1,1,1,1	4,4,4,4	3,2,2,2,2,2	4,4,4,4
P.I. 90763	III	1,1,1,1,1,1	4,4,4,4	1,1,1,1,1,1	4,4,4,4
P.I. 209332	III	1,1,1,2,2,1	4,4,4,4	2,2,2,2,2,2	4,4,4,4
P.I. 84751	IV	1,1,1,1,1,2	4,4,3,3	1,1,1,1,1,1	4,4,4,4

[a] Rating system: 1-no cysts; 2-1 to 10 cysts; 3-11 to 25 cysts; 4-26 or more cysts. Each number represents a single plant rating.
[b] Plant Introduction.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE; AND NORTH CAROLINA AGRICULTURAL EXPERIMENT STATION

DISEASES OF CROWN VETCH

J. H. Graham and K. E. Zeiders[1]

Crown vetch (Coronilla varia L.), a perennial legume, is grown to cover and stabilize the soil on highway slopes in Pennsylvania. It has been reported that this plant enjoyed "almost complete freedom from disease" (3). This was, indeed, a challenge to pathologists.

According to the "Index of Plant Diseases in the United States", issued by the United States Department of Agriculture (5), the root-knot nematode (Meloidogyne sp.) is the only organism reported to attack crown vetch. Chupp (2) reports that Cercospora rautensis C. Massal. has been noted on the plant in Italy, Hungary and Russia.

During June 1957, many fungi, including some pathogens, were found to be associated with crown vetch in central Pennsylvania. Of the fungi identified, Cercospora appeared to be the most damaging. Leaves, petioles, and stems were attacked severely, resulting in the death of plant parts. Lesions on leaves were round to oval, 1 to 2 mm long, and had brown centers with reddish-brown borders. Long, irregular, reddish-brown to chocolate-brown lesions developed on stems and petioles. The fungus was tentatively identified as C. rautensis.

Large, irregular, marginal lesions with light and dark brown concentric rings developed on leaves of crown vetch. Two species of Stemphylium were recovered. A large conidial type (av. 47 x 19 μ) borne on long conidiophores had smooth walls, was pointed at the apex, and appeared to be similar to S. solani Weber as reported on tomato (4) and lupine (6). The other type had small conidia (av. 24 x 16 μ) borne on short conidiophores. The conidia were smooth to minutely echinulate and generally were not pointed at the apex. The small conidia and conidiophores were on leaves and also on the beaks of perithecia on seed pods and pedicels. The perithecia contained old ascospores which resembled Pleospora herbarum (Pers. ex Fr.) Rab.

Anthracnose was prevalent on the leaves and stems of some plants. In morphology, the fungus ranged from Colletotrichum trifolii Bain & Essary to C. destructivum O'Gara, according to the descriptions. Following Arx (1), who recently monographed the genus, the fungus is identified as Glomerella cingulata (Ston.) Spauld. & Schrenk (C. gloeosporioides Penz.).

Species of the following genera of fungi were found on crown vetch but were not associated with any disease symptom: Fusarium, Rhizoctonia, Leptosphaeria, Ophiobolus, Phoma and Myrothecium.

It can be postulated that if crown vetch is grown extensively, the plant will have its share of diseases.

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STEMPHYLIUM SARCINAEFORME ON RED CLOVER IN URUGUAY[1]

W. E. Sackston[2]

Abstract

Stemphylium sarcinaeforme (Cav.) Wiltshire was found causing a leaf spot of red clover plants for the first time in Uruguay. S. botryosum Wallr., not previously reported in Uruguay, was found growing saprophytically on flax stems and other plant materials.

Red clover, Trifolium pratense, is being grown on an increasing scale as a pasture crop in Uruguay. Although such extremely destructive diseases as anthracnose and Sclerotinia root rot have not yet been reported, the leaf pathogens Uromyces trifolii, Cercospora zebrina, Dothidella trifolii, and Pseudopeziza sp. were included in a list of plant diseases in Uruguay, published in 1955 (1).

Leaf spots were numerous in mid-August of 1956 on the lower leaves of red clover plants in plots adjacent to the author's sunflower plots at La Estanzuela, Departamento Colonia. Urediospores of U. trifolii were present on some of the leaves, and apothecia of Pseudopeziza sp. developed on some small spots on leaves incubated in a moist chamber for 2 days.

Some of the larger spots, which averaged from 2 to 6 millimeters in diameter, and reached 10 millimeters where adjacent spots coalesced, had the alternating light and dark brown concentric zones characteristic of Stemphylium leaf spot. When leaves with such lesions were incubated in a moist chamber, spores of Stemphylium sp. developed profusely on them. The smooth conidia served to identify the fungus as Stemphylium sarcinaeforme, and to distinguish it from S. botryosum, which was found on dead flax stems and other tissues examined. Neither S. sarcinaeforme nor S. botryosum has previously been reported from Uruguay.

Single spore cultures of S. sarcinaeforme were established and were used in pathogenicity tests. The fungus was shown to be pathogenic by inoculations on excised red clover leaves collected in field plots and floated on water in Petri dishes, and on field-grown plants transferred to pots and brought into a screened shelter. Numerous lesions developed on the inoculated plants, and the pathogen was reisolated from them; uninoculated check plants remained free of the disease. Among the varieties included in the pot tests were La Estanzuela 116, the red clover variety commonly grown in the area of La Estanzuela, and eight varieties introduced under a program sponsored by the Food and Agriculture Organization of the United Nations. The numbers of plants of each variety tested were too small to determine any differences in varietal susceptibility to the disease.

Although S. sarcinaeforme has been shown to be pathogenic on red clover in Uruguay, the disease it causes probably will not become very important. Leaf spot diseases of forage crops do most damage in crops grown for seed, or cut for hay when the plants are fairly tall. Under such conditions, high humidities may prevail for long periods around the lower leaves, favoring sporulation and disease development. In Uruguay most of the clover is pastured by cattle and the plants are kept short, one of the better control measures for leaf diseases of forage plants.

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NATURAL INFECTION OF LADINO CLOVER BY THE RED CLOVER VEIN MOSAIC VIRUS

D. A. Roberts

Viruses recovered from naturally infected white clover, Trifolium repens L., are pea wilt virus, Marmor repens F. Johnson (7); pea mottle virus, Marmor efficiens F. Johnson (7); several strains of alfalfa mosaic virus, Marmor medicaginis Holmes (6, 8); tobacco streak virus, Annulus orae Holmes (3); and bean yellow mosaic virus (6). White clover has been infected experimentally with potato yellow dwarf virus, Aureogenus vastans (Holmes) Black (1); red clover vein mosaic virus, Marmor trifolii Holmes (4); New Zealand pea streak virus, Marmor iners Holmes (2); American pea streak virus (10); Wisconsin pea streak virus (5); and by what apparently was tobacco ringspot virus, Annulus tabaci Holmes (9).

Mosaic diseases of Ladino clover have been observed in all sections of New York State every year since 1951. The usual symptoms have been mottling with little leaf distortion and stunting of plant growth. Alfalfa mosaic virus was recovered from all mosaic diseased plants collected from 11 fields between 1951 and 1957.

A few Ladino clover plants in one field in northern New York in 1957 had moderately severe veinal mosaic symptoms that were indistinguishable from those induced experimentally in white clover (4). Diseased leaves were collected and were kept turgid for 1 day with petioles immersed in water in test tubes. These leaves were frozen overnight, thawed, and ground in a small amount of 0.01 M phosphate buffer, pH 7.0, with a mortar and pestle. Juice was rubbed on carborundum dusted leaves of Turkish tobacco, Nicotiana tabacum L.; Bountiful bean, Phaseolus vulgaris L.; Wando pea, Pisum sativum L.; sweet pea, Lathyrus odoratus L.; cowpea, Vigna sinensis Endl.; lupine, Lupinus sp.; broadbean, Vicia faba L.; and A & C cucumber, Cucumis sativus L. Inoculated leaves were rinsed with tap water. Three weeks after inoculation, infected pea plants were noticeably smaller than healthy non-inoculated ones. There was some chlorosis along veins of leaves of diseased plants, and lower leaves wilted, became bleached, and died. Infected pea plants died 5 weeks after inoculation. Diseased sweet pea plants showed moderately severe mosaic symptoms, and leaves began to die 4 weeks after inoculation. Plants died 7 weeks after inoculation. Infected broadbean plants were stunted and had mosaic; there was some systemic necrosis of leaves and petioles. Tobacco, bean, cowpea, lupine, and cucumber were insusceptible.

Ability to cause mosaic and death of sweet pea and inability to infect bean are characteristics that distinguish red clover vein mosaic virus from other viruses now known to infect white clover. Further, veinal chlorosis seems to be a diagnostic symptom in clovers infected by this virus. Symptoms produced in Ladino clover and in other susceptible legumes by one virus from Ladino clover in New York indicate that it is the red clover vein mosaic virus. This apparently is the first report of the natural occurrence of this virus in Ladino white clover.

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THE MOSAIC AND SIMILAR DISEASES OF SWEETPOTATO

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The recent report of a very severe mosaic of sweetpotato (Ipomoea batatas) in Georgia by Borders and Ratcliff (5) has stimulated the review of literature on sweetpotato virus diseases of this nature. The purpose of this paper is to record the results of this review and to familiarize research, extension and regulatory personnel with these diseases.

Early records of a sweetpotato mosaic in the United States came from Arkansas (8, 19, 20, 21), Florida (32, 33, 34), Kansas (1), Tennessee (26), and Texas, California and Mississippi (11). In all cases the records were on the variety Nancy Hall. The following description of the disease was given by Weber, 1923 (33):

> "Internodes decidedly shortened; thus the nodes bearing leaves are close together, affected plant showing a decided rosette appearance. The petioles are somewhat shorter and thicker than those of normal plants. The petiole at point of union with the blade is often flattened. The larger veins branch out into the blade abnormally suggesting fasciation and appear much wider and more prominent than the veins in normal leaves. The tissue between the veins is of a uniform green color, decidedly raised or sunken, forming pouch-like areas. There is no mottling, in the sense that leaves of cucumber and tobacco plants are mottled by mosaic. The blades are crinkled along the edges, curled backward and in severe cases malformed."

Definite transmission of the disease by workers at that time appears to have been questionable (11). Consequently, the virus nature of this mosaic is somewhat doubtful, especially in view of the established genetic nature of similar conditions (14).

On the other hand, the sweetpotato mosaic which recently was described from Georgia, and afterwards from Alabama (6) and possibly from California (4), appeared on the Porto Rico variety and was reported as being easily transmitted by grafting and carried by some unknown agent under field conditions (5). The following description of the mosaic was given by Borders and Ratcliff (5):

> "The diseased plants showed a mottling of the older leaves with raised blister-like dark green areas which gave the leaves a somewhat wrinkled appearance. Such leaves frequently were distorted in outline. Many of the small veins on some young leaves showed a yellowing very similar to that caused by the feathery mottle virus (Flavimacula ipomoeae) described by Doolittle and Harter. However, the mottling and blistering of the leaves were the predominant symptoms and appeared to be much more common than in the plants observed by Doolittle and Harter. The internodes of diseased plants were shortened and only a few very small fleshy roots were produced, whereas the uninfected plants in the Georgia fields produced normal yields."

There are numerous other relatively recent reports of virus and virus-like diseases of sweetpotato which may or may not be related to the sweetpotato mosaic described above. These reports include the following, in more or less chronological order:

1 -- From Cuba in 1934 Wellman (36) reported the occurrence on sweetpotato of the southern celery strain of ordinary cucumber mosaic virus. In 1935 (37) Wellman also reported the occurrence of this mosaic on sweetpotato in Florida (see description below).

2 -- From the Belgian Congo (2) in 1941 came a report of a disease apparently due to a virus. No description was available to the writer.

3 -- From East Africa in 1944 Hansford (9) described a disease of probable virus nature (see description below). No transmission experiments were reported. Later (10) Hansford reported similar symptoms on wild Ipomoea species.

4 -- From Tanganyika Territory Wallace (28) reported a virus disease on sweetpotato and six other species of Ipomoea, observed during a plant disease survey trip in 1945.

5 -- From the United States in 1945 Doolittle and Harter (7) described the feathery mot-

tle disease of sweetpotato, which they could transmit only by grafting. (See below for this description.) In 1954 mechanical and aphid transmission of this virus was reported by Webb and Larson (31), and different morning glory hosts of feathery mottle virus were reported by Webb (29). A morning glory indicator plant for the feathery mottle virus also was reported by Webb (30).

6 -- From the Lake Province of Tanganyika Territory in 1946 Miller (16) reported considerable crop loss from a sweetpotato virus disease.

7 -- From South Africa in 1947 McClean and Klesser (15) reported on the mottle-leaf disease of sweetpotato. The authors suggest that this disease may be similar to that described by Hansford (9) and Doolittle and Harter (7). (See description of this disease quoted below.)

8 -- From Kenya, East Africa, in 1949, Nattrass (17) reported that he was doubtful that a sweetpotato disease prevalent in Kenya was identical with that reported from the Belgian Congo (2). The Kenya disease was transmitted by grafting, producing symptoms of severe bunchy-top, vein-clearing and virtual absence of the internodes.

9 -- From East Africa in 1951, Sheffield (22) described symptoms (given below) of a virus disease of sweetpotatoes which was widely distributed and apparently identical with that described by Hansford in 1944 (9). She reported that spread was more rapid at medium (4000 to 5500 feet) than at high altitudes (above 6000 feet), and that there was no spread at all at low altitudes (3500 feet or below). In 1953 Sheffield (23) reported the isolation of three virus types, a very severe form, a moderately severe form, and a mild form. In 1953 she reported proof of the virus nature of this mosaic disease by graft transmission (24). In 1955 Sheffield (25) reported that of a number of insect families tested as vectors, only white flies transmitted the virus. She also reported the occurrence of another virus disease which she thought might be identical with the American feathery mottle, and stated that it was transmitted by Myzus persicae, in which it was non-persistent.

10 -- From Nyasaland in 1951 Lawrence (13) reported the occurrence of sweetpotato mosaic. No Description of this mosaic was available to the writer.

11 -- From the Ryukyu Islands in 1951 Summers (27) reported the occurrence of a dwarf disease caused by a virus (see description below).

12 -- From Ceylon in 1953 Newton and Peiris (18) reported a sweetpotato mosaic which was readily transmitted by inarch grafting.

13 -- From Puerto Rico in 1955 Adsuar (3) described a mosaic disease of sweetpotatoes (see description below), and reported transmission by core-grafting diseased cores into healthy roots and by cleft-grafting. The ordinary methods of juice inoculation were unsuccessful. He suggested a possible relationship with the East African disease described by Sheffield (24).

Wellman's (37) description of the mosaic on sweetpotato caused by the southern celery strain of ordinary cucumber mosaic follows:

> "...In the field a mild mottled pattern usually develops, which towards harvest time can hardly be recognized as of virus causation.
>
> "Sweet potato plants of the Porto Rico variety were inoculated by aphids and leaf-rubbing methods. The latter technique failed to produce infection, but aphids were successful in transmitting the virus. No primary lesions were produced, but vein clearing appeared in about 7 days after placing the aphids on the plants. The vein clearing finally disappeared and the next, younger, leaves to show infection were severely marked with a not very brilliant but distinct mottling characteristic of virus infection on this host. The plants were stunted and produced small roots."

The following description of the sweetpotato virus disease reported by Hansford (9) from East Africa is from the abstract in the Review of Applied Mycology:

> "The symptoms vary widely with the host variety. Some narrow-leaved varieties show severe stunting; the field has a yellow-green appearance, and individual plants grow very slowly, have small leaves, and show an excessive branching of the vines, with short internodes. The leaves generally display an indefinite mosaic of light and dark green areas, or, in other varieties, a light yellowish-green banding

along the veins. Older leaves frequently present bronzing of the entire leaf or of indefinite, scattered parts of the lamina. Enations were not observed. Broad-leaved varieties reveal more variation in leaf symptoms and reaction to infection. The Six Week variety shows almost complete infection, with pronounced mottling or mosaic of the leaves but little or no dwarfing; older leaves are slightly bronzed. Other broad-leaved varieties show mottling or mosaic or veinbanding, in some with distortion of the leaf shape, in others with leaf-crumpling, while parts of the leaf may be distinctly thick and tough. Most varieties show bronzing of the older leaves. On varieties which normally develop a red or purple pigment in the leaves and stems the symptoms are extremely obscure, particularly when dwarfing is absent."

Sheffield's (24) description of what she considered to be the same disease as that described by Hansford follows:

"The disease is of the mosaic type but the symptoms shown vary greatly. The most obvious are severe stunting and chlorosis. Internodes may be shortened, so that branching appears excessive. Leaves may be small, are sometimes distorted, are occasionally back-rolled at the extreme margin, and may be toughened and leathery. In varieties with much dissected leaves, they may be reduced to a strap-shape. A distinct vein-clearing may be shown, but this alone cannot be taken as a diagnostic feature since it commonly occurs in the young leaves of some varieties. A brilliant green and yellow mottle may be seen although this is unusual in the field, where mottling is more usually vague and indefinite. Probably only in those varieties which normally have some red pigment in their leaves, a green and bronze mottle may occur on the older leaves. Necrotic patches may occur on the leaves. These are seen also on the older leaves of some varieties when healthy and may be accentuated by infection.

"In some cases no obvious stunting is shown, the symptoms being confined to mottling, or one runner only may be affected or one branch only of one runner. It is probable that the stunted forms are primary infections (i.e., they arose from infected vines), the milder forms being secondary (or new) infections."

Doolittle and Harter's (7) description of feathery mottle of sweet potatoes from the United States follows:

" . . . In many plants the primary symptom is a pronounced yellowing along the veins of the younger leaves. This yellowing may outline many of the very small veins in a manner suggestive of frost patterns on a window pane . . ., or may affect only certain sections of the veins . . . On other plants, the first symptom may consist only of small, diffuse, yellow spots from 1 to 2 mm. in diameter . . . These spots, however, frequently occur together with some vein-clearing of the type mentioned. Symptoms of both types also are found on leaves produced after the plant has been infected for some time.

"In the later stages of the disease, the yellowing of the veins is often followed by the development of pale green areas that may occur as comparatively small spots or may be so large as to include one-third to one-half of the leaf . . . In such leaves the darker portions often are abnormally dark green and may show small, feathery areas of yellow along the veins. The leaves are sometimes slightly rugose and dwarfed but not much distorted. Infected plants commonly have shortened internodes, and there is a definite stunting of the plant in late stages of the disease . . . No necrotic symptoms have ever been noted on leaves, stems, or roots. The fleshy roots are smaller than those of healthy plants but infected plants in greenhouse always produce a fair number of medium-size storage roots.

"When mosaic plants grow slowly, the leaf symptoms are likely to be less pronounced than in rapidly growing plants. When growth is slow,

there usually are some leaves with only faint evidence of the disease but there is no general masking or extreme mildness of symptoms in the leaves of such plants. When plants are grown in the greenhouse at summer temperatures ranging from 75° to 105° F., the mottling is fairly pronounced. Temperatures as low as 60° to 65° F. do not suppress mosaic symptoms in the new growth."

Summers' (27) description of the dwarf disease of sweetpotatoes from the Ryukyu Islands follows:

"No edible sweet potatoes are produced on diseased vines. It is said that the incubation period is 70 to 120 days, so that many plants are affected without symptoms being apparent. Entire fields, even very large areas, have been observed in this condition. Visible symptoms consist of excessive proliferation of young shoots from leaf axils and dwarfing of subsequent growth. Little or no latex is present in diseased plants or roots."

McClean and Klesser's (15) description of the mottle-leaf disease of the sweetpotato follows (from abstract in Review of Applied Mycology):

"During 1946 a disease of the sweet potato, believed to be the same as that already reported from East Africa (by Hansford)* and the United States (by Doolittle and Harter)* was observed for the first time in the Nelspruit, Pretoria, Brits, and Rustenburg districts of the Transvaal. Half the cuttings from diseased plants died and the rest were severely stunted, growing to only one-third of the height of healthy plants. A characteristic symptom of both naturally and artificially infected plants is a network mottling of the mature leaves due to a yellowing along the course of the veins. In some naturally infected plants a mosaic mottling of mingled light and dark green areas without any yellowing was observed. Affected plants are generally pale in colour, with small, closely grouped leaves. The characteristic mottle was transmitted to healthy plants by grafting diseased shoots on to them. The disease is thought to be of virus origin and may multiply quickly in the field unless care is taken that stem and root cuttings are procured only from healthy plants."

Adsuar's (3) description of a mosaic of sweetpotato in Puerto Rico follows:

"In the field it appears as a distinct mosaic mottling of younger leaves. As the infected plant grows, the leaves become chlorotic and wrinkled. Sometimes these develop puckering in the form of raised green areas alternating with light-green areas. Affected plants are usually stunted. Frequently, however, the above-mentioned symptoms assume a milder manifestation and close scrutiny is necessary to detect infected plants in the field. Laboratory transmission by tuber or cleft-grafting usually produces very severe symptoms in susceptible varieties in from 3 weeks to 1 month, consisting of pronounced vein-clearing, chlorosis, wrinkling of leaves, and stunting, followed in many cases by death of the plant."

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FORECASTING LATE BLIGHT OF POTATO IN AROOSTOOK COUNTY, MAINE, IN 1957

Reiner Bonde, R. A. Hyre, and Barbara Johnson

Abstract

The estimated mean foliage blight of potato, due to Phytophthora infestans, was about 1 percent. This small amount of blight was quite accurately reflected by both the rainfall-temperature and the relative humidity-temperature methods of predicting blight.

INTRODUCTION

A forecast and warning service was conducted for late blight of potato, due to Phytophthora infestans, in Aroostook County, Maine for the third successive year. A study of different methods of forecasting blight and of some of the variables encountered was continued. The amount of blight present in the county toward the end of the season was then determined for comparison with the "expected" amount. The blight forecasts were made at Presque Isle by the senior author.

METHODS

The method used for making the forecasts was similar to that used in 1956[1]. This year rainfall and temperature data were forwarded at 5-day intervals by collaborators of the United States Weather Bureau for the four stations of Houlton, Presque Isle, Caribou, and Fort Kent. Blight was forecast after 10 consecutive days when both rainfall and temperature were favorable and the weather forecast at that time was for continued blight-favorable weather. Rainfall was considered favorable when the 10-day total was 1.2 inches or more. Temperature was considered favorable when the 5-day average was less than 78° F. Any day was considered unfavorable, however, if the minimum temperature was less than 45° F. On the basis of previous experience the disease can be expected 1 or 2 weeks after it is forecast.

For comparison, blight-favorable periods were determined also from relative humidity and temperature data by locating a hygrothermograph in a shelter which was placed on the ground near potato plots at Aroostook Farm, Presque Isle. Periods were considered favorable for blight when the relative humidity was 90 percent or more and the temperature was 45° to 53° F for at least 16 consecutive hours, 54° to 59° for 13 hours, or 60° to 80° for 10 hours.

During the last week of August the amount of foliage blight present in the county was estimated. For this purpose a total of 357 fields was graded by the method of Horsfall and Barratt[2].

RESULTS

Figure 1 shows an analysis of rainfall and temperature data for Houlton, Presque Isle, Caribou, and Fort Kent; and the blight-favorable periods at Aroostook Farm (Presque Isle) as determined from relative humidity-temperature data. Table 1 gives the amount of blight present in the county during the last week of August. By this time some growers had already started killing the tops.

The great variation in the number of "blight-favorable" days at different locations in the county is readily apparent from Figure 1. This number varied from a minimum of 12 at Houlton to 41 at Caribou for the growing season of June 10 through August 31. The 41 favorable days at Caribou were 25 more than the 16 favorable days at Presque Isle, which is about 14 air miles away. These data emphasize the value of obtaining weather records from several stations instead of just one station when forecasting blight for a relatively large area.

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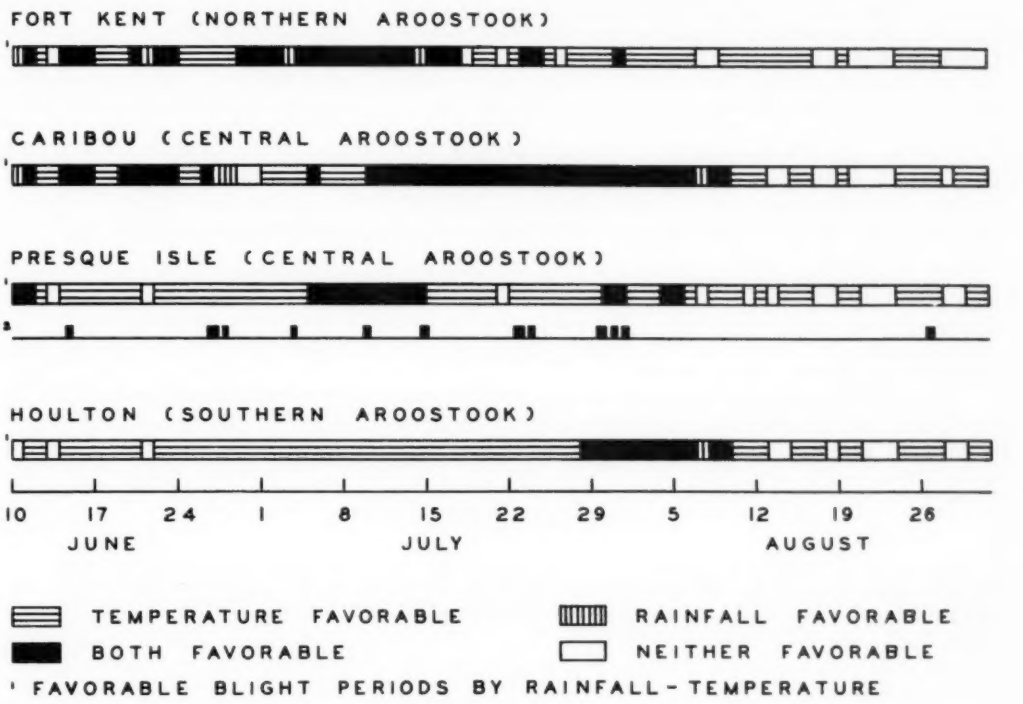
FORT KENT (NORTHERN AROOSTOOK)
CARIBOU (CENTRAL AROOSTOOK)
PRESQUE ISLE (CENTRAL AROOSTOOK)
HOULTON (SOUTHERN AROOSTOOK)
10 17 24 1 8 15 22 29 5 12 19 26
JUNE
JULY
AUGUST
TEMPERATURE FAVORABLE
RAINFALL FAVORABLE
BOTH FAVORABLE
NEITHER FAVORABLE
1 FAVORABLE BLIGHT PERIODS BY RAINFALL-TEMPERATURE CRITERIA (SEE TEXT FOR CRITERIA).
2 FAVORABLE BLIGHT PERIODS BY RELATIVE HUMIDITY-TEMPERATURE CRITERIA.

FIGURE 1. Analysis of weather data and the occurrence of late blight of potato in Aroostook County, Maine in 1957. The first reports of late blight were received the last 2 weeks of July.

Table 1. Extent of potato late blight infection in Aroostook County, Maine, as estimated August 26-30, 1957.

Location	Number of fields	Percent of foliage blight
Northern Aroostook (Fort Kent)	53	0.3
Central Aroostook (Presque Isle and Caribou)	237	0.9
Southern Aroostook (Houlton)	67	1.5
Total	357	0.9

From Table 1 it is seen that very little late blight was present in 1957 -- about 1 percent foliage blight. An occasional field had considerable blight, but many fields had none at all and numerous fields showed a trace of infection that apparently was weeks old with no recent infection on the subsequent growth of foliage. These older infections probably occurred during the last 2 weeks of July, following blight-favorable weather, when the first reports of blight were received from the central (Caribou, Presque Isle) and northern (Fort Kent) parts of the county.

By late August somewhat more blight was found in the southern part (Houlton) of the county than elsewhere, probably because the blight-favorable weather occurred later in the season at Houlton than elsewhere, excepting at Caribou. Also, the more scattered, smaller fields at Houlton might indicate a somewhat less thorough spray program than was employed farther north.

The lack of infection of potato cull piles undoubtedly was an important factor in the small amount of blight in the county this year. The senior author inspected about 50 cull piles in the spring without finding any blight. The good spraying program carried out by most of the potato growers in Maine also was a big factor in the relatively small amount of late blight infection in Aroostook County in 1957.

The initial occurrence of blight and the total amount for the season agreed quite well with that "expected" for the forecast method, except possibly in the Caribou area. The heavier rainfall there might have been quite local since it was not reflected in the Presque Isle data. The relatively few "favorable" periods for blight at Presque Isle, according to the relative humidity-temperature method, were in agreement with the small amount of blight present.

MAINE AGRICULTURAL EXPERIMENT STATION, ORONO, MAINE; CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, NEWARK, DELAWARE

FURTHER STUDIES WITH STREPTOMYCIN FOR WILDFIRE CONTROL IN BURLEY TOBACCO PLANT BEDS, NORTH CAROLINA, 1957

Luther Shaw and G. B. Lucas[1]

Summary

Excellent control of wildfire (Pseudomonas tabaci) of burley tobacco resulted from four weekly spray applications of either streptomycin sulfate or streptomycin nitrate. Best control was obtained at a concentration of 200 ppm as compared with 100 ppm or with 3-4-50 Bordeaux mixture drench. Either two or three spray applications of streptomycin sulfate at a concentration of 400 ppm, beginning 1 week after inoculation of the plants, gave good control of the disease.

INTRODUCTION

Wildfire control studies in burley tobacco plant beds were continued at the Mountain Research Station, Waynesville, North Carolina, along the same general lines followed in 1955 and 1956 (1, 2). A brief report of the methods employed and the results obtained in the 1957 studies are presented.

MATERIALS AND METHODS

The materials and methods employed in 1957 were essentially the same as those employed previously (2). Plant bed areas were treated with methyl bromide for weed control and were seeded with the Kentucky 16 variety. Plants were inoculated by spraying with a water suspension of the wildfire parasite (P. tabaci) at twilight. The inoculum was prepared from 32-hour-old cultures of the bacterium incubated at room temperature. The plants were in an advanced two-leaf stage when inoculated. The schedule of treatments (Table 1) was begun 12 hours after inoculation. Subsequent applications were made at 1-week intervals. Spray treatments were applied to give thorough coverage of the plants. Hence, as the plants increased in size the volume of spray required for coverage increased, resulting in a range in rate of application from 3 to 5 gallons per 100 square yards. Disease readings were taken 28 days following the first applications and 7 days following the last applications. From these readings disease indices were calculated by the same procedure followed in 1956 (2).

RESULTS

Weather conditions were generally favorable for wildfire development during the course of this experiment. The disease appeared in the check plots and in some of the treated plots 10 days following inoculation. Stand reduction was evident in the check plots although detailed counts of this damage were not made.

The disease index values (Table 1) indicate that all of the bactericidal treatments, with the exception of one application of streptomycin sulfate made 1 week following inoculation, resulted in significant control of the disease. It is of particular interest that both streptomycin sulfate and streptomycin nitrate gave significantly better disease control at a concentration of 200 ppm than they did at a concentration of 100 ppm in this test. At the 200 ppm concentration these materials gave significantly better control of the disease than did the 3-4-50 Bordeaux mixture drench. The eradicative properties of streptomycin sulfate on P. tabaci following infection of the plants was further demonstrated in this test with highly significant disease control from two and three applications at a concentration of 400 ppm beginning 1 week following inoculation. The results of these tests suggest that a concentration of 100 ppm of streptomycin is not sufficient to adequately control wildfire in burley tobacco plant beds when weather conditions are favorable for development of the disease.

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Table 1. Wildfire control with varied concentration and schedules of application of streptomycin sprays in burley tobacco plant beds, North Carolina, 1957.

Treatment and concentration	Method of application	Rate per 100 sq. yds. (gallons)	Number of applications	Disease index June 4
Streptomycin sulfate 100 ppm	Spray	3-5	4	11.4*
Streptomycin sulfate 200 ppm	"	3-5	4	5.5**
Streptomycin sulfate 100 ppm + maneb 1/2 lbs./100 gals.	"	3-5	4	10.8*
Streptomycin sulfate 200 ppm + maneb 1/2 lbs./100 gals.	"	3-5	4	6.0**
Streptomycin nitrate 100 ppm	"	3-5	4	11.1*
Streptomycin nitrate 200 ppm	"	3-5	4	5.5**
None (check)				15.3
Streptomycin sulfate 400 ppm	"	3-5	1[a]	14.1
Streptomycin sulfate 400 ppm	"	3-5	2[a]	8.8**
Streptomycin sulfate 400 ppm	"	3-5	3[a]	6.8**
Bordeaux mixture 3-4-50	Drench	25	2	10.3*
None (check)				16.8
L.S.D. .05				4.1
.01				5.5
C.V. (%)				28

[a] The first application was made 1 week after the beds were inoculated with P. tabaci.
* Significant at the 5% level.
** Significant at the 1% level.

A trace of blue mold (Peronospora tabacina) was found in some of the plots on the date the wildfire readings were made; however, it was not severe enough to measure the effectiveness of any of the treatments in the control of this disease. Anthracnose (Colletotrichum sp.) did not appear in these beds.

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RHIZOCTONIA BUD ROT OF STRAWBERRY

Stephen Wilhelm[1]

During the past few years serious losses in strawberry have occurred throughout the central and southern coastal growing areas of California from a bud rot caused by Rhizoctonia solani. In all important details this disease was similar to that described by Demaree (1), but differed from the Rhizoctonia disease of strawberry described by Zeller (5). Zeller reported on Rhizoctonia clones which were primarily root pathogens of strawberry (Marshall variety) and which apparently did not attack crown tissues. The severe symptoms of wilting and occasional death of Rhizoctonia-affected plants reported by Zeller may have been caused by Verticillium albo-atrum which was reported present in a considerable number of affected plants. The Rhizoctonia bud rot of strawberry described in this report has aspects similar to the crown bud rots of hops and alfalfa described by Jones (3) and Erwin (2), respectively, and attributed to the same fungus.

In strawberry, the so-called terminal bud is actually the apex of a short compressed shoot called "crown", on which many buds closely appressed to each other occur at each node. Normally, most of these buds are latent. Buds first to grow after winter typically become inflorescences, those developing later become runners or side crowns. The disease described in this report is typically one of the crown apex, which for convenience is here called the terminal bud.

SYMPTOMS

Small groups to large areas of strawberry plants scattered throughout many fields have been observed in which the terminal buds had been killed during the winter and spring rainy seasons. The normally vigorous 2-year-old plants presented a shabby appearance, because much of the terminal crown growth had been killed, and the only live growth was from spindly lateral crowns arising considerably below the soil surface (Fig. 1 B).

In addition to being unproductive because of the loss of all flower buds, such plants frequently were poorly anchored in the soil. This may reflect the fact that roots initiated among the dead crown tissues and old leaf bases frequently were killed before making contact with the soil. Frequently and typically following protracted rain, petioles of old leaves were rotted at their bases and remained attached to the plant by the frayed remnants of the vascular strands.

Newly set plants which may be carrying Rhizoctonia, or which may be set in infested soil, have either failed to grow or, after the loss of the terminal bud, have tardily produced spindly side crowns from latent lateral buds. Leaflets of such crown growth frequently are disfigured and deeply serrated, the petioles etiolated and distinctly reddened (Fig. 1 A). By midsummer severely affected plants with numerous spindly crowns resembled witches' brooms and appeared more like wild strawberry types than cultivated varieties. Less severely affected plants frequently recovered sufficiently to appear perfectly normal, but commonly still carried Rhizoctonia.

These symptoms suggested that an organism dependent upon high moisture for pathogenesis, active during low temperatures when plant growth was relatively slow, was causing the disease.

ISOLATION OF RHIZOCTONIA

Isolations made during the past 2 years from crown tissues of numerous diseased plants have consistently yielded the fungus Rhizoctonia solani, most frequently rapidly growing, dark sclerotial types. Unfolding leaflets with water-soaked areas, petioles showing slightly sunken, reddish-brown lesions, and lateral buds from below the ground, frequently imbedded in decaying tissues, commonly yielded the fungus. Adventitious roots emerging from among the old leaf bases frequently, but not always, yielded R. solani. From these, Cylindrocarpon radicicola and Pyrenochaeta terrestris were isolated as frequently as Rhizoctonia.

Isolations were made by plating diseased tissues, after soaking them in 10 percent commercial Clorox, on 2 percent water agar to which barley straw, sterilized with propylene

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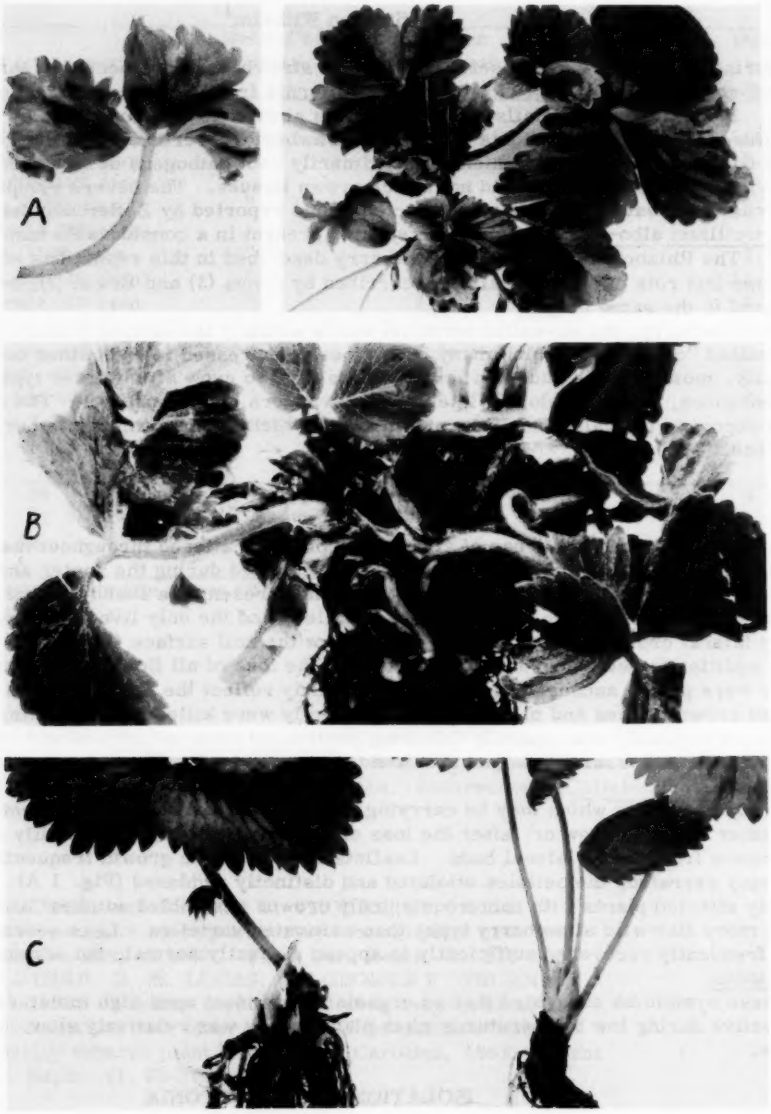

FIGURE 1. A. A first year Shasta strawberry showing disfigured and deeply serrated central leaflets due to Rhizoctonia injury to buds. The inset shows a disfigured leaf in greater detail. B. A second year Shasta strawberry showing extensive terminal bud rot caused by Rhizoctonia solani. C. Left -- A Shasta inoculated with Rhizoctonia solani after 20 days. Right -- A Rhizoctonia-free check.

oxide, had been added. Within 48 hours after plating, the Rhizoctonia fungus frequently had grown at least half the distance across the Petri dish, and could be obtained in pure culture by transferring hyphal tips. A rather consistent fungus flora and fauna were present in the Rhizoctonia-infected tissues. Two kinds of mycophagous mites frequently appeared on the plates, as did also certain "free-living" nematodes. Fusarium oxysporum, F. roseum, Cylindrocarpon radicicola, Chaetomium species, Pyrenochaeta terrestris, Gliocladium roseum, Macrophomina phaseoli, and Zythia fragariae with its ascigerous stage, Gnomonia fructicola, were the most commonly encountered associated fungi. Some of these, that is, F. terrestris, M. phaseoli, and G. fructicola, are recognized pathogens and may have aggravated the Rhizoctonia disease.

PROOF OF PATHOGENICITY OF RHIZOCTONIA

Inoculations with pure cultures established R. solani as the causal organism of this disease. For inoculum, the fungus was grown on autoclaved pieces of dried tomato stems about 2 inches long until they were thoroughly colonized. One such unit of inoculum was placed alongside each dormant strawberry plant (Shasta variety) as it was potted into a 6-inch pot. The potting soil had been rendered free of plant pathogens by previous fumigation with chloropicrin. Typically, 20 plants were inoculated with each isolate of Rhizoctonia tested. Three isolates of Rhizoctonia from strawberry crown tissue and two from roots of tomato, these latter isolated by Mr. Donald Ford, produced typical symptoms of the disease as described above for newly set plants. In each test the terminal crown tissues were killed within 15 days after inoculation. Spindly, reddened shoots bearing disfigured leaves arising from latent lateral buds usually appeared within 30 days (Fig. 1 C). In no test did all 20 of the inoculated plants become diseased; a few always escaped. Upon culture, however, these escaped plants in every instance yielded Rhizoctonia from the surface crown tissues. Perhaps environmental conditions and factors of nutrition favored the escaped plants more than they did the parasite, thus deterring active parasitism. Leach (4), in studies of damping off caused by Rhizoctonia, showed that seedlings would commonly escape if their relative rate of growth was greater than that of the parasite. For strawberry, the degree of maturity (dormancy) attained at the time of digging in the nursery, the degree to which the chilling requirement had been satisfied, and the conditions of storage all may affect the vigor with which a given individual may begin growth.

NURSERY STOCKS MAY BE INFECTED WITH RHIZOCTONIA

Specific scattered outbreaks of the Rhizoctonia disease in commercial fields, fumigated with chloropicrin before planting for the control of Verticillium wilt, prompted the investigation of nursery stocks as possible carriers of the pathogen. A survey of the major nurseries of California confirmed the suspicion that planting stocks may be infected. Not only was it possible to detect diseased plants in the nurseries by inspection, but Rhizoctonia was isolated from nearly all collections of material suspected of being diseased. The isolates were similar in cultural characteristics to those obtained from commercial fields.

Control measures, in addition to soil treatments, should center around methods of rendering infected planting stocks free from Rhizoctonia, since one of the principles of plant pathology is that "Disease production is more certain in instances of seed or planting-unit-transmission of the pathogen because of the simultaneous distribution of pathogen and host, the contiguity of pathogen and susceptible host tissues, and the likelihood of the presence of a virulent strain of the pathogen." Control methods are being studied.

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RELATIVE RESISTANCE OF SOME STRAWBERRY SPECIES, VARIETIES, AND SELECTIONS TO POWDERY MILDEW AT SAANICHTON, BRITISH COLUMBIA[1]

W. R. Orchard and M.C.J. van Adrichem

Powdery mildew (Sphaerotheca macularis (Wallr. ex Fries) W.B. Cooke) (= S. humuli (DC.) Burr.) is one of the more serious diseases of strawberry in British Columbia, and no information is available upon the relative resistance of species, varieties and selections; consequently, the disease ratings were established for 7 species, 32 varieties and 33 selections in August 1957 at Saanichton.

Table 1 gives the mildew ratings according to the system applied by Darrow et al. (2) and Miller and Waldow (3).

Of the seven species rated, three were found free of mildew infection. In the species F. glauca an interesting feature shown by the data is the high resistance of the Northern British Columbia and Yukon selections compared with the relatively high susceptibility of the local Vancouver Island selection.

Auchincruive No. 6 was also found to be free of the pathogen. This variety is a selfed seedling from No. 52, which is known as Frith, found in Northern Scotland and used in breeding for red stele resistance. From the selections, it can be seen that there is no clear cut line in inheritance to resistance or susceptibility to powdery mildew. Quite often a cross shows more resistance, rates the same, or is more susceptible than either parent.

It should also be pointed out that in relation to Verticillium wilt resistance Wilhelm (4) found that a hybrid may have a value beyond the paternal limits. This was also observed by Baker (1) in relation to the fruit characters in strawberry.

Of the 75 species, varieties, and selections rated, 4 showed no sign of powdery mildew infection; 16 can be rated as resistant, although a trace of mildew could be found. The remainder can be classed as susceptible.

In order to compare the results obtained at Beltsville by Darrow and others (2) and at Corvallis by Miller and Waldo (3), the varieties at those locations and the similar varieties rated at Saanichton are presented in Table 2.

As shown in Table 2, the ratings at Beltsville and at Corvallis do not differ to any great extent. The Saanichton ratings, however, do show differences. Sparkle, Climax and British Sovereign were more susceptible at Saanichton, while Marshall was more resistant. The other ratings at Saanichton do not show a marked difference from the Beltsville or Corvallis ratings.

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[1]Contribution No. 1640 from the Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario

Table 1. Powdery mildew resistance of some strawberry species, varieties and selections, at Saanichton, British Columbia, 1957.

Species, variety or selection	Rating[a]	Species, variety or selection	Rating[a]
Fragaria glauca (Muncho Lake, Yukon)	10	Aberdeen	7
F. glauca (Watson Lake, B. C.)	10	Cambridge Rival	7
F. orientalis (S. E. Asia)	10	Jucunda	7
F. chiloensis (Vancouver Island)	8	Sierra	7
F. ovalis (Vancouver Island)	8	3205-3 (Sparkle x Fairland)	7
F. chiloensis (Oregon)	7	4608-3 (Magoon x Marshall)	7
F. glauca (Vancouver Island)	7	526 (Red Crop x Magoon)	7
F. virginiana (S. Ontario)	7	467-1(6) (Temple x Campbell)	7
F. bracteata (Vancouver Island)	6	59	7
F. moschata (Bot. Gard. Moscow, U. S. S. R.)	6	P.P.S. 6	7
		P.P.S. 5	7
Auchincruive No. 6	10		
		Agassiz	6
Cambridge Sentry	9	Cambridge Early	6
Huxley	9	Climax	6
Madame Moutot	9	Deutsch Evern	6
Marshall	9	Macherauchs Frühernte	6
Siletz	9	Madame Lefeber	6
Ydun	9	Magoon	6
Perle de Prague x Magoon 1	9	Northwest	6
Perle de Prague x Magoon 4	9	Perle de Prague	6
4607-5 (Magoon x Premier)	9	Regina	6
4502-8 (Paxton x Magoon)	9	Climax x Magoon 5	6
4501-30 (British Sovereign x Magoon)	9	Climax x Temple	6
4502-18 x Climax 1	9	Md. 683	6
4502-18 x Climax 3	9	Sparkle x Temple	6
4502-18 x Climax 4	9	55	6
P.P.S. 7	9	4607-9 (Magoon x Premier)	6
P.P.S. 4	9	4607-1 (Magoon x Premier)	6
		P.P.S. 1	6
Fairfax	8	P.P.S. 2	6
MacIntyre	8	P.P.S. 3	6
Oberschlesien	8		
Premier	8	British Sovereign	5
Sparkle	8	Red Crop	5
Talisman	8	Scarlet	5
Temple	8	Stelemaster	5
Perle de Prague x Magoon 2	8		
Perle de Prague x Magoon 3	8		
4606-9 (Magoon x Marshall)	8		
4607-3 (Magoon x Premier)	8		
5214 (Red Crop x Magoon)	8		
4502-18 x Climax 2	8		

[a] 10 = no mildew; 9 = trace of mildew but no leaf rolling; 8 = small amount of mildew with slight leaf rolling; 7 = leaves considerably mottled but little or no leaf rolling; 6 = much leaf rolling with some injury to lower surface (reddening and beginning necrosis); 5 = much leaf rolling accompanied by severe necrosis (injury severe enough to cause loss of leaves).

Table 2. Comparison of resistance to powdery mildew in strawberry varieties at Beltsville, Corvallis, and Saanichton.

Variety	Beltsville rating	Corvallis rating	Saanichton rating
Sparkle	10	9.5*	8
Marshall	8	7.7*	9
Climax	9	9	6
Fairfax	8	--	8
Premier	8	--	8
Stelemaster	6	5	5
Siletz	--	9 *	9
Northwest	--	5.7*	6
Sierra	--	7.5*	7
British Sovereign	--	7.5*	5

* Average for 2 or 3 years.

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BOTANY AND PLANT PATHOLOGY DIVISION, SCIENCE SERVICE, CANADA DEPARTMENT OF AGRICULTURE

RELATION OF PRECIPITATION TO MAT FORMATION BY THE OAK WILT FUNGUS IN NORTH CAROLINA

John S. Boyce, Jr.[1]

Mat formation by the wilt fungus Ceratocystis fagacearum on felled oaks in western North Carolina differed markedly in 1956 and 1957. Table 1 shows that 9 percent of the trees cut in the late summer of 1955 bore mats during the following January through April, whereas 79 percent of the similarly treated 1956 trees had mats the following January through April.

Furthermore, on the 1 out of 11 trees cut in 1955 that was found to be mat bearing early in 1956, only one mat 6 square inches in area formed during the above months. In contrast, 114 mats totaling 1,936 square inches formed on 23 of the 29 trees cut in 1956 and observed for mats early in 1957. All of the mats had cracked the overlying bark so that they were exposed to insects. The size ranges of the trees cut during both years were roughly comparable. The wilt fungus was isolated from each tree observed during this study.

One factor which influenced rate of drying of the felled trees and subsequent mat formation was the amount of precipitation during the period August through December of each year. For this period in 1956, rainfall was 76 percent greater than it was in 1955 (Table 1). Total precipitation was comparable for the winter and early spring months of 1956 and 1957, being 13.7 inches and 15.5 inches respectively. Thus, much more late winter and spring mat formation occurred on summer-felled wilt trees following a wet autumm than after a comparatively dry one. The average monthly temperatures for the August to April periods of each year after cutting were similar. This suggests that, after a wet fall, trees that have been summer-felled and sprayed with the bark intact (1, 2) as a method of oak wilt control should be sprayed again early the following year. For example, a spray applied about March 15 in western North Carolina will reduce insect infestation of mats during the spring months.

Table 1. Mat formation by the wilt fungus on felled oaks in North Carolina.

Year	Total rainfall August-December[a] (inches)	Total stems observed (number)	Stems with mats next January-April (number)	(percent)
1955	8.9	11	1	9
1956	15.7	29	23	79

[a] At Asheville, North Carolina.

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NEW OR UNUSUAL RECORDS OF PLANT DISEASE OCCURRENCE

MASTIGOSPORIUM ALBUM RIESS ON ALOPECURUS PRATENSIS IN NOVA SCOTIA

By A. G. Johnson[1]

While viewing the beautiful scenery at Peggy's Cove, Nova Scotia, in July 1954 it was noted that all the plants in a patch of meadow foxtail (Alopecurus pratensis L.) were severely attacked by a leaf-spotting and leaf-blighting organism, apparently a fungus, as minute white spore masses were evident in the center of some of the older spots. Subsequent microscopic examination showed the characteristic fungus, Mastigosporium album Riess.

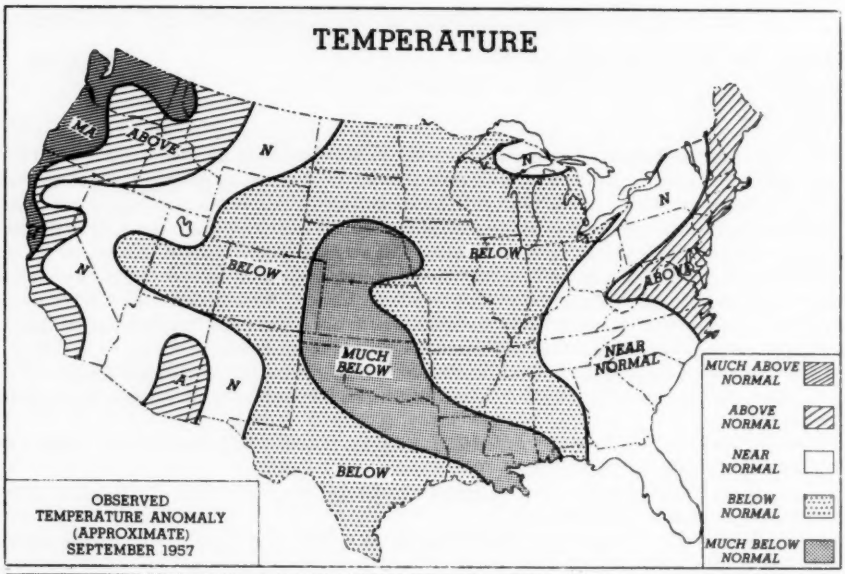
The characteristics of the leaf spots on the younger leaves agree perfectly with those described by Ross[2] from specimens collected near Ithaca, New York. In addition, the older leaves of the Nova Scotia material had been so severely attacked that they were all dead. It was on these leaves, however, that the best spore masses were found.

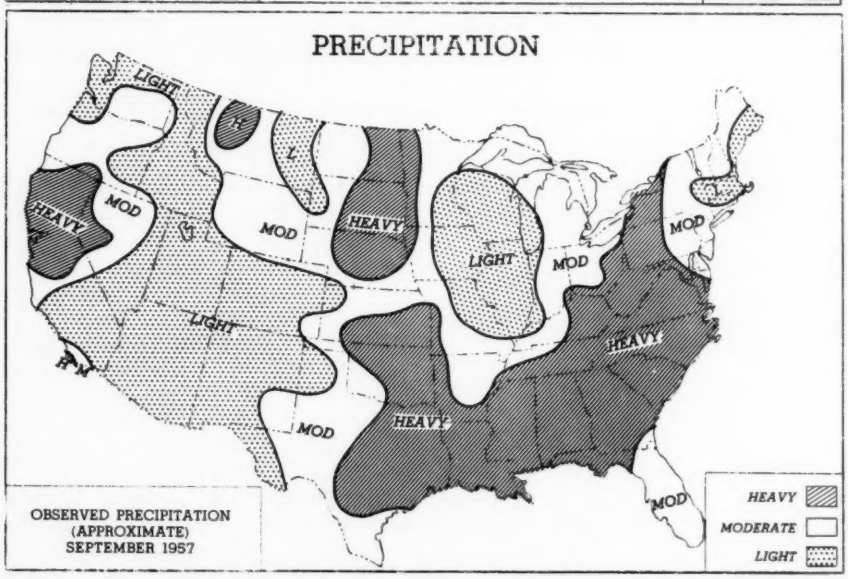
This is the second report for this fungus in North America. Specimens have been deposited in National Fungus Collections, United States Department of Agriculture, Beltsville, Maryland.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

[1]Plant Pathologist, Collaborator, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[2]Ross, J. P. 1954. Mastigosporium album Riess on Alopecurus pratensis in New York. Plant Dis. Reptr. 38: 607-608.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

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(See also PDR 35: 121-122; 36: 214, 494)

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